Significance of the Praesystolic Murmur.

BY

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I propose detaining the Faculty a few minutes with some remarks upon a point of analytical diagnosis in connection with disease of the heart.

SIGNIFICANCE OF THE PRAESYSTOLIC MURMUR.

Some years ago (in 1867), a case came under my observation, which made me question the explanation, which I had adopted on the authority of Barth and Roger, Walshe and Flint, of the sound which was described first by Fauvel in 1843, and then by Grisolle as the praesystolic murmur, afterwards by Dr. Gairdner of Edinburgh as the auricular systolic murmur, and by Dr. Austin Flint, Sr., as the mitral direct murmur.

These authorities claimed that this sound was heard just *preceding* the ventricular contraction, and was caused by the systole of the auricle forcing the blood into the ventricle, through a diseased and contracted auriculo-ventricular orifice.

The case was of a man 64 years of age, of grossly intemperate habits, who came to the Baltimore Infirmary with symptoms of advanced heart disease—great dyspnoea, a small contracted pulse, heart much hypertrophied, with a murmur of a rasping character, heard loudest between the second and third ribs at the base, not extending up the carotids, but down toward the base, and completely obliterating the second sound of the heart. The murmur was audible after the apex-beat and the systole of the ventricle, and was followed by the pause of the heart. The first sound of the heart was normal.

The diagnosis seemed clear and unmistakable, and was recorded

as insufficiency of the aortic orifice, by means of which the arterial blood was forced back into the left ventricle.

The heart being obliged to contract more frequently, so as to supply the organism with the proper quantity of blood, the muscular walls had become from this extra work enlarged, and thus, as the great dullness over the praecordial region indicated, there was compensating hypertrophy.

For a time this increased size and force of the central organ accomplished the usual rôle of the heart, but the disease increasing, the individual suffered more and more, until shortly after admission into the hospital he died, suffering intensely from cardiac apnoea.

The post-mortem showed ætheromatous degeneration in the aorta above the semilunar valves extending to the sacks of Valsalva, and causing adhesion of one of the semilunar pouches of the aortic orifice to the wall, so binding it down that that portion of the orifice was unprotected. Thus, at the rebound of the artery the blood was partly sent back into the ventricle. The second sound could not be produced, and the insufficiency of the valve was evident.

Thus far the diagnosis was correct, but on examining the mitral orifice we found, to our surprise, that it was reduced by thickening at its base to about the size of one-quarter of an inch in diameter. Yet during life, there was no abnormal sound preceding or during the ventricular systole. With such a contraction of the left auriculo-ventricular orifice, ought we not to have had a decided praesystolic murmur? The whole heart, auricle and ventricle, was enlarged and increased in force, and yet there was no murmur produced from the passage of the blood through an orifice so reduced in size! I could not help questioning the received opinion as to the significance of the so-called mitral direct murmur. As it is a physical sound, heard at a particular period of the heart's action, the physical cause which was said to produce it being present, it ought to have been heard, but it was not.

Since this case I have been much interested in the articles that have appeared at different times, discussing the mode of production of this sound. I have had several cases in which I have heard this sound immediately preceding the impulse of the heart,

or apex beat and the first sound of the heart. Although I have tried, I have never been able to get a post-mortem demonstration of the cause of sound. Yet I have insisted in my clinical teachings that I believed it was not caused by the passage of blood through the mitral orifice, but by intra-ventricular disease—not by stenosis of the orifice, but by abnormal friction within the ventricle.

Before I give in detail the grounds, physiological and pathological, upon which I base this opinion, I would refer first to cases which have been recently reported by others, where, notwithstanding there was found the post-mortem lesion of great contraction of the orifice, yet there was discoverable during life no praesystolic murmur.

I will first give the facts bearing upon the point in discussion, and then deduce my conclusions as to the explanation of those facts.

This abnormal sound is heard over a limited area, recognised as the mitral area. The position of its audition is limited ordinarily above by the third rib, and below by the middle of the sixth intercostal space. Its rhythm or relation to the several physiological acts is easily recognised. During the period of the heart action, we have the ventricular systole synchronous with the apex beat and the first sound, the ventricular diastole synchronous with the second sound ; next, the period of cardiac rest, as it is called, but during which the blood is pouring into the cavities of the heart, on the right side through the venae cavae and coronary veins, on the left through the pulmonary veins ; and lastly, the systole of the auricle, which is quick and sudden, and consumes two-tenths of the time of the heart's action. This last period in the heart's labor is apparently the moment when we hear this murmur. It is distinguishable immediately before the first sound, and has been regarded as produced by the column of blood passing through the constricted mitral orifice.

We have mentioned our own case, and now briefly give those which have been observed by others.

Hope, as far back as 1842, reports a case of a man named Christian Anderson, where the mitral orifice was so contracted that it would only admit the little finger, yet there was no murmur during life preceding the first sound. In his report he adds, "I

have frequently known a contraction of the mitral orifice to the size of only two or three lines to occasion little or no murmur."

Mr. Prescott Hewett has described a case in which the mitral orifice was reduced to the size of a quill, and during lifetime no signs of diseased heart were exhibited.

Dr. James R. Leaming reports the following case in the New York *Medical Record:*

"Mrs. B——, 23 years of age, native of New York, widow, called Dr. S——, in April, 1869, for advice as to cardiac trouble and swelled feet. The Doctor found on examination a systolic murmur over the base of the heart, more distinct over the aortic valves, gradually disappearing to the right in the course of the aorta; there was also a diastolic murmur.

"*Diagnosis.*—Aortic obstruction and aortic regurgitation with hypertrophy of left ventricle. There were also casts in the urine, and albumen. She became dropsical, her condition gradually grew worse, and she died in September last.

"I saw the case with Dr. S——, in May, and found no different conditions than those already discovered. *There was no mitral murmur of any kind.* The specimens here presented show Bright's small kidney of advanced disease. The heart is hypertrophied mostly in the left ventricle; the aortic valve is thickened at the base of the curtains; shortened to incompetency—so far agreeing with the diagnosis. But the mitral valve presents the most notable feature. There was no sign of disease of this valve during life, and yet it is damaged in a very peculiar manner. It is thickened by lymph deposit; its color white, opaque; the edges of the curtain are adherent, and the orifice is narrowed down till it will barely admit the tip of the index finger; and the whole valve extends down into the cavity of the ventricle like a funnel. The chordæ tendineæ were shortened and thickened by lymph deposits, and the musculi papillares were thickened and lengthened. But everything was symmetrical, viz., the funnel-like condition of the valve, the hypertrophy of the cardiac walls, of the musculi papillares, and of the columnæ carneæ. With perfect conditions for producing a *mitral direct murmur*, it was absent."

Dr. Stokes, in his work on diseases of heart and aorta, relates two cases of extreme contraction of the mitral orifice found after

death, but where during life there had been no murmur audible, even to his practised ear.

Dr. Waters, of Edinburgh, in the second edition of his work on diseases of the chest, just published, in the sixth chapter, writes of the praesystolic murmur. While he gives the received opinion, that it is caused by stenosis of the mitral orifice, he details cases in which he shows there was no connection between the sound and the lesion. He speaks of them as exceptional cases. His first case is where he heard a loud systolic as well as a praesystolic murmur. At the autopsy there was found insufficiency and slight contraction of the mitral orifice. In his second case there was no praesystolic murmur whatever, although the autopsy showed a constricted mitral orifice, only admitting the tip of the index finger. Next follow the details of four cases of extreme contraction of the mitral orifice, where during life there was no praesystolic murmur audible. He candidly adds: "I have given you instances sufficient to prove that great constriction of the mitral orifice may exist without there being any murmur produced by the passage of the blood from the auricle into the ventricle, and therefore that you must not look for a mitral diastolic or praesystolic as a constant sign of obstructive mitral disease. My belief is that this murmur is far more frequently absent than present, even when there is great obstruction at the mitral orifice." Dr. Waters accounts for the presence or absence of this murmur as depending on the greater or less vigor with which the auricle contracts.

We have now given ten cases beside our own where examination after death showed the lesion which ought to have produced the sound during life, but did not do so. Dr. Waters says truly, "Its absence is no proof that obstruction, even to an extreme degree, does not exist;" and Balfour, an unwilling witness, confirms this view by saying, "This murmur is not always audible when its cause is present." Further, Dr. Flint records three cases in which the murmur was loud and clear, and yet the mitral valves were found at the post-mortem to be normal, but there was insufficiency of the aortic valves. Thus we have eleven cases of the lesion without the murmur, and three cases of murmur without the lesion!

These two sets of facts we consider valuable, as contributing to

prove that the praesystolic murmur is not due to stenosis of the mitral orifice.

Before we get to what we believe to be the true explanation of this sound, let us look for a moment at the anatomical and physiological reasons against the usually received view. The sound is not a smooth, short one, but it is a rough, blubbering sound, often perceptible through thick clothing, and has been called "the praesystolic purring," (Niemeyer). It is also prolonged, according to Flint and Balfour, up to the actual moment when we hear the apex-beat and the first sound, showing considerable force, whereas the muscular development of the auricle is very slight. Again, the time occupied by the murmur usually far exceeds two-tenths of a second, which, Marey tells us, is the time consumed by the auricular systole. The exceedingly wide orifice, between the auricle and the ventricle, measuring in circumference from four to four and a half inches, extending nearly through the whole septum, shows us the facility with which nature intended the blood should pass into the ventricle through the auricles, and the absence of any necessity of any but feeble muscular power to assist it.

The physiological objections to the old theory seem to be very well founded. We must bear in mind that the auricles are not first filled with blood and then contracted to force the blood into the ventricles, but during the diastole the blood flows onward, without obstruction and without force, except the feeble, gentle current of the venous system, and both cavities are nearly, if not quite full before the systole commences, the feeble muscular fibres of the auricles beginning to contract, and the contraction going right on to the powerful fibres of the ventricles. In fact, the ventricles are full before the auricles give their first impetus to the current that forces open the aortic valves and over them into the aorta. So true is this, that Chauveau has shown that the systole of the auricles is not immediately necessary to the preformance of the circulation. He exhausted the contractility of the auricles, nevertheless the ventricles continued to act and keep up the circulation without their aid. Even Harvey speaks of the harmony of the auricular and ventricular systole, the two concurring in such wise that but one motion is apparent. He compares it to a set of wheels acting upon each other when they

appear to move simultaneously. We do not wonder at this action when we look closely into the histological structure of the walls of the heart, and find them composed of innumerable muscular fibres arranged like two balls of twine, each with a cavity in its centre, and both completely enveloped in a third ball. The free inosculation of the fibres, a peculiarity of the muscular structure of the heart, and the absence, according to Robin, of the sarcolumna, favor the complete systole of the heart.

When the auricles use their slight force the ventricles are nearly full, and the force is conveyed against the whole volume of blood, and is expended far beyond the mitral orifice. Before the auricular systole commences the blood has flown into the ventricles, and as demonstrated by Baumgarten of Germany, and by Doctor Halford, the flaps of the auriculo-ventricular valve rise on the surface of the liquid and come in contact. As the systole extends to the ventricle, they are brought forcibly together, and their tension gives us the valvular element, the real cause of the first sound of the heart.

We hold that the contraction of the auricles is a secondary one, incapable of producing the prolonged rough sound recognised as the praesystolic murmur.

Dr. Flint, Sr., thinks that the auricular force is sufficient to throw the curtains into vibration as the blood passes through the slit-like aperture, and thus produce the mitral direct murmur; but at that time there is no resistance to the force of the auricular systole, there is nothing to prevent their yielding. With due deference to so high an authority, we cannot consider this explanation as tenable.

How then do we account for the sound which is heard and recognised as the praesystolic murmur? We believe it to be produced by endocardial causes within the ventricle, during the powerful ventricular systole, and not before the systole, although apparently so. The impulse of the heart and the forcible closure of the valves do not consume the whole of the time of the systole of the ventricle. They are produced at the *culmination* of the systole—the end.

What occurs during the systole of the ventricles? The blood is pushed both onward and backward with great force, equal,

according to Hales, to 51.5 pounds, which will raise blood in a tube connected with the aorta 7 feet 6 inches. We know if the onward current meets with asperities at the aortic orifice we hear a rasping murmur; so may it not be with the backward current, which passes between the cordae tendinae, and closes with a sharp sound the curtains of the mitral valve? In reading over carefully Dr. Balfour's elaborate article in the *Edinburgh Medical Journal*, we were struck with the fact that in every case where he found the praesystolic murmur, there was also thickening from some cause of the free edge of the mitral curtains, or shortening and induration of the chordae tendinae. These lesions could have of themselves caused the murmur independently of the mitral contraction.

The abnormal friction, then, of the backward current of the blood against these rugosities up to the point of complete adjustment of the valves, and a twisting of the apex against the ribs, can give us precisely the sound we would expect. It appears to be praesystolic, because the sound strikes the ear before the completion of the systole, as evidenced by the impulse and the first sound.

Waters and the other authorities agree that the praesystolic is generally associated with a true systolic murmur. In chronic disease of the heart we know that isolation is the exception and complication the rule.

Several authors speak of a fact in relation to this murmur which is to us hardly consistent with the mitral stenosis theory. It is that this sound may exist at one time and not at another (Waters), may disappear temporarily, or even permanently (Balfour. Dr. Gowers, in *London Practitioner* for Dec., 1873, gives four cases where it was heard in the recumbent position, but disappeared in the erect position. Stokes also speaks of its singular behavior in not being sometimes audible.

Does this occur in aortic stricture and its characteristic sound? No matter what the position of the body, the blood must be forced through the contracted orifice, and we ought to have the physical result of the extra friction. We can easily account for insufficiency murmurs becoming inaudible as the space increases, because there is less friction. A smaller orifice with the same force will

make a louder noise; but in obstructive lesions the orifice through which the blood is forced becomes smaller as the disease advances, and the murmur should be louder. Inside the ventricle the area is much larger, and we can see how it is much more probable that the blood-current may not so come in contact with the rugosities as to cause the *frémissment cataire* which Thauler considers characteristic of the praesystolic.

If stenosis of the orifice is the physical cause of its production, then the intensity of the sound ought to be in proportion to the lessening of the size of the orifice. Again, how can the sound disappear? If we have given an orifice, a fluid of a fixed consistence and a force, the sound produced in one case must always be produced in a similar case.

Flint tells us that praesystolic continues up to the occurrence of the succeeding first or systolic sound. Again, that it is more intense at its end than at its beginning. All these facts add probability to our conclusions as to the nature of this morbid sound, that stenosis of the mitral orifice is not the cause of its production. We claim with Dr. Leaming, who we believe was the first, in a very suggestive paper in the New York *Journal of Medicine* (1868), to propose the ventricular origin of the so-called mitral direct murmur—"That this murmur is only heard when the mitral valve is much diseased; and that the thickening and irregularity of the mitral valve, with the irregularly hypertrophied ventricular walls and columnae carnae, are the physical causes of this sound, the blubbering character being produced by the irregular tension of the chords; and the murmur is formed at the commencement of the ventricular systole, and not by the auricular systole."